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Please delete claims 2 and 3 from the application.

Claims 6 through 14 have previously been withdrawn. Accordingly, the application remains with amended claims 1, 4 and 5.

#### REMARKS

Claim 1 of this application has now been restricted to include the limitations of previous claims 1 and 2, i.e. to include that the SEG is obtained from flaxseed and has a purity of at least 95%.

The claims on file in this application are restricted to a method for treating hypertension or for reducing or preventing development of elevated blood pressure.

With regard to the cited prior art, the Clark et al. patent is for use of SDG in lupus nephritis which is an auto-immune disease. Nowhere in the patent is there any description that SDG produces a fall in the blood pressure. Lupus varies greatly in severity from mild to severe where other organs such as kidney, heart, lungs and brain are damaged. Lupus nephritis is inflammation of the kidney in persons suffering from lupus.

SDG is a compound having a purity of greater than 95% isolated from flaxseed. This patent application describes its hypotensive effects both in normotensive and hypertensive rats and its mechanisms of hypotensive effects.

The Talom et al. paper describes the effect of flaxseed, specifically n-3 polyunsaturated fatty acids (n-3 PUFAs), on the vascular reactivity in rats. They have not studied the pure compound (SDG) isolated from flaxseed. Flaxseed contains SDG, p-coumaric acid glucoside and ferulic acid glucoside and hydroxy-methylglutaric acid, besides n-3 PUFAs. Using flaxseed does not provide any clue as to which component has hypotensive effects. SDG, p-coumaric acid and ferulic acid have antioxidant activity and hydroxy-methylglutaric acid has hypocholesterolemic activity. Also flaxseed contains cyanogenic glucosides. Cyanogenic glucosides have an adverse effect on health. Flaxseed also contains phytate which will inhibit the absorption of calcium.



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In other words, use of flaxseed does not provide any suggestion as to which component is active as hypotensive agent. Also because of cyanogenic glucosides and phytate, chronic consumption of flaxseed has deleterious effects. SDG because of its purity will not have these side effects.

Talom et al. showed that endothelium-dependent vasorelaxation is impaired in spontaneously hypertensive rat (SHR) and that high flaxseed diet (20% flaxseed in diet) improves responsiveness of SHR to both acetylcholine and bradykinin without changes in arterial pressure. These authors also conclude that endothelial dysfunction is not essential to the development of hypertension. Their paper suggests that flaxseed diet improved the endothelial dysfunction.

Endothelial dysfunction is a feature of vascular diseases and is not the cause of the disease. Improvement of endothelial cell function does not cure the disease or cause of the disease. It would only reduce/prevent the complications. Endothelial cell dysfunction is associated with numerous conditions including diabetes, hypertension, hypercholesterolemia, cigarette smoking, aging, coronary artery disease, congestive heart failure, ischemia-reperfusion such as angioplasty and bypass, eclampsia and syndrome X, endotoxemia, pulmonary hypertension, posttransplantation and reckettsial infection.

Endothelial dysfunction in these diseases has been suggested to be due to oxidative stress subsequent to the disease. Various interventions such as lipid lowering therapy, ACE inhibitors, exercise, antioxidants, smoking cessation, glucose control and blood pressure control have been successful in improving endothelial function. Talom et al. showed that flax diet-induced improvement in endothelial function appear to involve a pressure-independent mechanism since increased responsiveness to acetylcholine and bradykinin occurred despite lack of changes in blood pressure. It has been postulated that elevated blood pressure could, through alterations in structure or function, trigger a decrease in release of relaxing factor from endothelium as a secondary process leading to endothelial dysfunction.

Talom et al. suggest that flaxseed contributes to an increased responsiveness to vasodilator influences and that this effect is greater in a hypertensive animal relative to its normotensive counterpart. This does not mean that flaxseed is hypotensive. This only means that flaxseed improves the endothelial function. Hypertension produces endothelial dysfunction as pointed out above. Improvement in endothelial function with flaxseed may be due to its antioxidant activity. There are data which suggest that oxidative stress which is

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increased in hypertension could damage the endothelium and can produce endothelial cell dysfunction.

Antioxidants have been shown to improve endothelial cell function in atherosclerosis, hypercholesterolemia, diabetes and cigarette smokers.

Acetylcholine and bradykinin which Talom et al. have used to show that flaxseed diet increases their vasodilator responses in hypertensive rats are normally and regularly used to test endothelial dysfunction. Improvement in endothelial cell function by any agent does not mean that the agent is hypotensive.

From the above explanation it will be seen that there is nothing to suggest that Talom et al. in any way recognized that SDG as a substantially pure compound could be an effective reagent for treating hypertension and reducing or preventing development of elevated blood pressure. Furthermore, there is nothing from the Talom et al. paper to suggest that flaxseed can lower blood pressure, only that it improves endothelial cell function.

